Chapter 11
Pediatric TBI

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Abstract Pediatric traumatic brain injury (TBI) is the most common cause of acquired brain injury in childhood and leads to neuropathological, neurocognitive, and neurobehavioral morbidities. Unfortunately, these morbidities frequently persist and impact day-to-day long-term outcomes including adaptive behavior, educational attainment, employment, and quality of life. Relative risk of poor outcome is influenced by a variety of individual (e.g., age at injury, cognitive reserve), socioeconomic, and injury-related (e.g., duration of posttraumatic amnesia) factors. Certain sequelae, particularly neurocognitive deficits, have been tied to neuroanatomical abnormalities, most often via volumetric and diffusion tensor magnetic resonance imaging. While application of cognitive rehabilitation approaches has lagged behind that found in adult TBI, recent research has targeted neurocognitive function after pediatric TBI, with modest success albeit limited evidence of far transfer of effects. Neuroimaging has been very seldom used to assess treatment-related changes in brain system integrity after pediatric TBI but provides a robust opportunity to elucidate the mechanisms underlying response to intervention in this population.

Keywords Pediatric · Acquired brain injury · Neuroimaging · Sequelae Adjustment

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J. DeLuca et al. (eds.), *Cognitive Rehabilitation and Neuroimaging*,
https://doi.org/10.1007/978-3-030-48382-1_11

Pediatric TBI Background

Epidemiology of Pediatric TBI

Traumatic brain injury (TBI) occurs when the usual function of the brain is disrupted by a bump, blow, or jolt to the head or by a penetrating injury (Marr & Coronado, 2004). TBI is the most common cause of acquired brain injury in childhood and is a leading cause of death and disability in children and adolescents (Faul, Xu, Wald, & Coronado, 2010). Although prevalence and incidence rate reports vary across sources, recent estimates provided by the United States Centers for Disease Control and Prevention (CDC) indicate that an estimated 640,000 emergency department visits and 18,000 hospitalizations in 2013 were due to TBI (Taylor, Bell, Breiding, & Xu, 2017). Compared to estimates from a decade prior (i.e., 2002–2006), this is a slight increase in emergency department visits, but a significant decrease in hospitalizations, down from roughly 60,000 (Faul, Wald, Xu, & Coronado, 2010). This may not reflect a true increase in the incidence of TBI, but rather a growing public health concern about TBI (particularly mild TBI, or concussion) and corresponding state legislation dictating healthcare provider clearance to return to play for youth athletes.

Rates of pediatric TBI and associated emergency department visits and hospitalizations are not distributed randomly across the population or across injury mechanism; rather, they vary based on both demographic and injury-related factors. Boys are about twice as likely to sustain a TBI as girls across all ages. Incidence rates are particularly high among very young children (i.e., ≤4 years old; 1,035 per 100,000), largely due to injuries sustained in falls or as a consequence of inflicted, non-accidental head trauma. Non-accidental trauma (e.g., injury due to abuse or assault) also contributes to the relatively greater mortality rate in this age group. Among older children and adolescents, falls remain a common mechanism of injury, as do motor vehicle accidents and other transportation-related injuries (e.g., bicycle accidents), although sport- and recreation-related injuries are the single most common mechanism. Older adolescents (i.e., ages 15–19 years) have a higher rate of hospitalization and death from TBI, given the frequency of injuries sustained in motor vehicle collision, which tend to be disproportionately more severe than injury by other mechanisms.

Socioeconomic status and race are also linked to variability in incidence and prevalence rates (especially for more severe injuries), hospitalizations, and deaths from TBI arising from motor vehicle crashes; specifically, among young children (i.e., ages 0–9 years), those who are Black are more likely to be hospitalized or die from their injuries, as are those of lower socioeconomic status. Black, Hispanic, and Native American children are also more likely than White children to sustain a severe TBI and have a higher mortality rate (Centers for Disease Control and Prevention, 2011; Falcone Jr, Martin, Brown, & Garcia, 2008; Howard, Joseph, & Natale, 2005; Linton & Kim, 2014). Pre-injury emotional and behavioral functioning may also play a role in the incidence of TBI; children with pre-injury psychiatric

disorders (e.g., ADHD, anxiety), for example, are more likely to sustain a TBI than are children with no psychiatric history (Bloom et al., 2001).

Rates of pediatric TBI also vary based on injury severity. Though imperfect, the most common method of classification of TBI severity is the Glasgow Coma Scale (Teasdale & Jennett, 1974), which integrates information about the extent of motor response, verbal response, and eye-opening to rate level of consciousness. Specific criteria are outlined in Table 11.1, with total scores ranging from 3 to 15; scores from 3 to 8 are traditionally classified as severe, scores from 9 to 12 as moderate, and scores from 13 to 15 as mild. Some injuries are described as "complicated mild," which indicates GCS score of 13–15 accompanied by significant abnormalities on neuroimaging (typically computed tomography [CT] scans) conducted at the time of injury. Often additional criteria are considered when assessing TBI severity; for instance, assessment using GCS alone can lead to questionable classification when a child is particularly young, or when a child or adolescent requires medical sedation. In these cases, classification metrics such as the pediatric version of the GCS (pGCS), duration of loss of consciousness, or duration of posttraumatic amnesia can provide important additional information. Criteria for the pGCS are annotated(*) in Table 11.1, and a summary of classification criteria is provided in Table 11.2.

Table 11.1 Glasgow Coma Scale

Category	Response	Score	Description
Motor response	No response	1	Flaccid
	Extension	2	Decerebrate posturing
	Abnormal flexion	3	Decorticate posturing
	Normal flexion	4	Normal flexor response—Gross withdrawal from pain
	Localizing	5	Localized response to pain (*in pGCS, withdraws to touch)
	Obey commands	6	Follows basic commands (*in pGCS, moves spontaneously/purposefully)
Verbal response	No response	1	No vocalization
	Incomprehensible/ sounds	2	Vocalization, but no discernible words (*in pGCS, moans in response to pain)
	Inappropriate speech	3	Intelligible speech, but no coherent thought process (*in pGCS, cries in response to pain)
	Confused speech	4	Responsive to individual questions, but responses remain disoriented (*in pGCS, irritable cries)
	Oriented	5	Normal orientation to person, place, and time (*in pGCS, coos/babbles)
Eye opening	No response	1	None, and not due to ocular swelling
	To pressure/pain	2	Opening when pain applied to chest or limbs
	To sounds	3	Opening in response to speech/shouting
	Spontaneous	4	Spontaneous opening

*Indicates score descriptor differences specific to the Pediatric application of the Glasgow Coma Scale

Table 11.2 Summary of severity classifications

	TBI severity		
Criteria	Mild[a]	Moderate	Severe
GCS	13–15	9–12	3–8
pGCS	13–15	9–12	3–8
Loss of consciousness	<30 min	30 min to 24 h	>24 h
Duration of posttraumatic amnesia	<24 h	24 h to 7 days	>7 days

[a]Complicated–mild TBI fits the listed criteria for mild TBI, accompanied by trauma-related abnormalities on neuroimaging

Regardless of method, the vast majority of TBIs in childhood are classified as mild. Though somewhat variable across studies, epidemiological estimates indicate that between 70 and 90% of TBI-related visits to the emergency department are for injuries eventually classified as mild TBI (Cassidy et al., 2004; Faul et al., 2010; Rivara et al., 2012). TBI places a significant financial burden on the public healthcare system. One recent study estimated that, within the first 3 months after an injury, related costs exceeded $1,000 for mild injuries, $4,300 for moderate injuries, and $7,200 for severe injuries. Though costs are clearly higher for individual severe TBI relative to mild TBI, population-level costs for mild TBI were estimated to approach $700 million within 3 months after injury because of the high frequency of mild TBI, while costs for moderate and severe TBI were estimated at $81 and $175 million, respectively (Graves, Rivara, & Vavilala, 2015).

Fortunately, overall mortality rates are on the decline, with roughly 1,500 deaths attributed to TBI in 2013 (Taylor et al., 2017) relative to an average of 6,100 between 2002 and 2006 (Faul et al., 2010). As mortality from TBI decreases, long-term morbidities are increasingly recognized and have been the focus of concerted research efforts. At least 145,000 children and adolescents are thought to be living with persistent and significant social, behavioral, physical, or cognitive sequelae from TBI (Zaloshnja, Miller, Langlois, & Selassie, 2008). These long-term morbidities vary widely by injury severity and associated neuropathological and pathophysiological characteristics of the injury.

Neuropathology and Pathophysiology of Pediatric TBI

Pediatric TBI can result in a variety of neuropathological changes. Some are a direct result of the impact on the brain itself, whereas others are a consequence of a cascade of neurochemical processes that follow the impact. Some changes occur immediately upon impact, while others emerge or evolve over the days, weeks, and months following the injury (Farkas & Povlishock, 2007; Giza & Hovda, 2001; Povlishock & Katz, 2005; Toledo et al., 2012). Structural neuropathological changes are often discussed as either primary or secondary consequences of TBI. Primary injuries occur as a direct result of the force to the head and include things like skull

fracture, lacerations and contusions, and shear or strain injury to brain tissue or vasculature. In contrast, secondary injuries come about indirectly; some examples include brain swelling or edema and increased intracranial pressure. Swelling can also cause a reduction in blood flow, leading to hypoxic or ischemic injury.

Primary injuries reflect both the biomechanics and the physics of injury. In many cases, these injuries occur due to the properties of acceleration–deceleration. Upon impact, a child's skull may abruptly stop, but the brain tissue does not. In addition to a possible skull fracture, this acceleration–deceleration can cause tearing of blood vessels and shear injury to nerve fibers. These injuries contribute to evolving hemorrhage and diffuse axonal injury, respectively (Yeates, 2010a). Focal lesions may also occur; they disproportionately occur in the anterior frontal and temporal brain regions (Wilde et al., 2005) partially because of the greater likelihood of sustaining a TBI to the front of the head, and partially due to the presence of bony prominences in the anterior and middle fossa of the skull (Bigler, 2007).

In many cases, secondary injuries that arise over time are the most life threatening, including brain swelling, cerebral edema, and associated increased intracranial pressure. When swelling occurs, the normal balance of blood volume, cortical tissue, and cerebrospinal fluid is disrupted. When cerebral blood flow is diminished, blood volume can increase, further contributing to elevated intracranial pressure. At their worst, swelling and elevated intracranial pressure can cause herniation of the brain and even death; at milder levels, hypoxic or ischemic injury can occur, exacerbating any associated excitotoxic damage (Toledo et al., 2012; Yeates, 2010a).

Other neuropathological changes occur due to neuroinflammatory and metabolic cascades during and after the acute injury period. After TBI, inflammation can lead to an excess of microglia and microphages, as well as reactive astrogliosis (Kelley, Lifshitz, & Povlishock, 2007; Shultz, Bao, et al., 2012, Shultz, MacFabe, Foley, Taylor, & Cain, 2012). Additionally, TBI can lead to the excessive production of free radicals and excitatory amino acids, which can disrupt cell metabolism and lead to cell death (Hunter, Wilde, Tong, & Holshouser, 2012). Several studies utilizing magnetic resonance spectroscopy have noted reduced *N*-acetylaspartate (NAA), even in the absence of visible injury on conventional neuroimaging. This likely reflects the neuronal dysfunction and neuronal loss that occurs acutely after TBI. Choline, on the other hand, is often elevated due to shear injury to myelin and cell membranes. Glutamate is another excitatory amino acid that can be released into extracellular space after TBI. This also contributes to neuronal death, particularly in regions with a large number of glutamate receptors such as the hippocampus (Wilde et al., 2007). TBI can also disrupt normal cellular calcium homeostasis; when elevated, circulating calcium can worsen the effects of hypoxic or ischemic injury.

Although some of the acute neuropathological changes that occur after TBI are expected to abate over time, others are more lasting. Specifically, while metabolic and neurochemical changes are temporary, cortical thinning, cerebral atrophy, and associated ventricular enlargement can be persistent. Diffuse axonal injury and white matter degeneration persist, especially in cases of severe TBI. Recent longitudinal research has provided a clearer appreciation of the temporal dynamics of

white matter pathology after TBI (e.g., Dennis, Babikian, Giza, Thompson, & Asarnow, 2017; Wilde, Ayoub, Bigler, Chu, Hunter, Wu, et al., 2012). White matter injury is most often assessed using diffusion tensor imaging (DTI), which quantifies the extent of the restriction of diffusion of water along axons. During DTI, information about water diffusion is collected across a variety of spatial directions and modeled as an ellipsoid. The most common DTI metric used to convey white matter organization is fractional anisotropy (FA), which is the ratio of the extent of diffusion along the three main axes. Ranging from 0 to 1, an FA value of 0 indicates entirely isotropic diffusion, while an FA value of 1 indicates entirely anisotropic diffusion (i.e., perfect diffusion along the white matter tract). Thus, lower FA values generally suggest compromised white matter integrity. Close to the time of injury (i.e., within 3 months), however, FA tends to increase due to the impact of brain swelling on the restriction of water diffusivity. In contrast, farther from the time of injury, FA tends to decrease as swelling recedes and damage to white matter can be detected more clearly.

Typical Cognitive Profile and Impact on Everyday Functioning

The typical neurocognitive and neurobehavioral consequences of TBI involve domains that correspond to the brain systems most vulnerable to traumatic injury, particularly the anterior frontal and temporal cortical regions and white matter pathways. Subcortical structures can also be affected, leading to difficulties with autonomic regulation and motor coordination. The assessment of the neurocognitive and behavioral sequelae of TBI demands an appreciation for normal developmental processes and of how recovery or the emergence of deficits occurs in both a developmental and environmental context that has implications for both prognosis and intervention.

After mild TBI (especially a single, uncomplicated mild TBI), the development of long-term cognitive deficits is highly unlikely (Babikian et al., 2011; Babikian & Asarnow, 2009; Bijur, Haslum, & Golding, 1990; Brosseau-Lachaine, Gagnon, Forget, & Faubert, 2008; Carroll et al., 2004; Hung et al., 2014; Yeates, 2010b; Yeates et al., 1999, 2009). More likely, falling outside of the realm of cognitive deficits, nearly a third of children and adolescents who sustain a mild TBI endorse behavioral or psychological symptoms that persist beyond 4 weeks. In some cases, these symptoms reflect pre-injury health or psychosocial factors rather than the injury itself, perhaps exacerbated by inaccurate expectations a child or family may have about the consequences of mild TBI (Covassin, Moran, & Wilhelm, 2013; Eisenberg, Andrea, Meehan, & Mannix, 2013).

Overall, neurocognitive and behavioral sequelae of TBI are far more common in moderate to severe TBI. The typical pattern of deficits following moderate to severe injury is outlined below.

Neurocognitive Functioning

A wealth of literature outlines associations between TBI and outcomes in various neurocognitive domains. Early research efforts relied on global assessment of intellectual functioning and found that children with TBI performed more poorly on IQ measures relative to either healthy control or orthopedic injury control groups. Global measures have been criticized, however, as they can obscure more nuanced strengths and weaknesses. For instance, children with TBI often perform more poorly on nonverbal intelligence tasks, or those with a speed-based component, relative to verbal intelligence measures (Babikian & Asarnow, 2009). Thus, more recent investigations have focused on more specific aspects of neurocognitive functioning.

The most common and specific neurocognitive changes following TBI include deficits in nonverbal skills, attention, memory, processing speed, and executive functions (e.g., cognitive flexibility, working memory, planning, organization, self-regulation; Babikian & Asarnow, 2009; Babikian, Merkley, Savage, Giza, & Levin, 2015). Although overt aphasias are rare following the acute injury period, subtler language deficits can persist, particularly in pragmatic domains (Yeates, 2010a).

Executive dysfunction is particularly common, perhaps due to the contribution of prefrontal and cingulate regions to these skills and the vulnerability of such regions to injury. These deficits can persist, even up to 10 years post-injury in cases of severe TBI (Beauchamp et al., 2011; Mangeot, Armstrong, Colvin, Yeates, & Taylor, 2002). In terms of prevalence, one study found that roughly 20% of children with severe TBI displayed symptoms consistent with attention-deficit/hyperactivity disorder (ADHD), compared to only 4% of children with orthopedic injury. When premorbid history of attention problems was taken into account, rates of post-injury clinically significant symptoms of attention problems on the CBCL increased to 82% for children with severe TBI, relative to 42% of children with orthopedic injury (Yeates et al., 2005).

Psychosocial Adjustment/Psychiatric Functioning

Emotional and behavioral disorders can also emerge following TBI and can also contribute to overall impairment, decreased quality of life, and poor functional independence. Behavior problems and mood disorders such as depression or anxiety are common, more so in children and adolescents with severe injuries (Schwartz et al., 2003), affecting up to nearly two-thirds of those with severe TBI (Bloom et al., 2001). Children and adolescents with TBI are more likely than their healthy peers to display disruptive behaviors, conduct problems, and emotional distress (Anderson, Catroppa, Haritou, Morse, & Rosenfeld, 2005) and are at increased risk of engaging in risky lifestyle behaviors such as substance use and self-harm (Babikian et al., 2015). Unfortunately, although many of the cognitive deficits that occur after TBI

subside over time, emotional and behavioral symptoms are more persistent, and may even worsen (Fay et al., 2009), especially in the context of poor family functioning or discord (Yeates et al., 2001).

Psychosocial morbidities are also common after TBI. Children and adolescents may become socially withdrawn from peer groups or become reluctant to participate in previously enjoyed activities (Bedell & Dumas, 2004). They may display poor social skills, lower self-esteem about social skills, reduced social participation and peer interaction, and greater loneliness and isolation. Their poor social outcomes may be related to impairment in basic social skills, poor self-regulation during social interactions, and difficulties with social problem-solving. Studies examining predictors of social adjustment have noted that the developmental timing of the injury, injury severity and location, and the impact of the TBI on basic cognitive and social-cognitive skills are important considerations (Rosema, Crowe, & Anderson, 2012; Yeates et al., 2007). Furthermore, the quality and pervasiveness of social difficulties can change over time. In a sense, children can "grow into" social deficits as they transition to adolescence, likely due to the increasing complexity of social relationships (Tonks et al., 2009; Turkstra, 2000; Turkstra, McDonald, & DePompei, 2001). Although neurocognitive deficits may play a role in difficulties navigating the complex social world of adolescence, other aspects of social information processing contribute more to the variability in social adjustment observed after TBI. Theory of mind, or the ability to make inferences about the internal or mental states of others, can be particularly challenging for children following severe TBI and accounts for significant variance in social adjustment, above and beyond performance on measures of executive function or processing speed (Robinson, Fountain-Zaragoza, et al., 2014).

Adaptive Functioning

Traumatic brain injury can also impact day-to-day adaptive functioning. Following TBI, particularly severe injuries, children and adolescents are at increased risk of deficits in adaptive functioning, including reduced social participation, leisure skills, and social adjustment (Shultz et al., 2016). Deficits in communication, daily living skills, and other functional behaviors have also been documented (Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008; Fay et al., 2009; Stancin et al., 2002; Taylor et al., 1999, 2002), particularly when family functioning is poor (Taylor et al., 1999). Unfortunately, these types of deficits also tend to persist and contribute to worse long-term outcomes (Cameto, Levine, & Wagner, 2004; Koskiniemi, Kyykkä, Nybo, & Jarho, 1995; Nybo, Sainio, & Müller, 2004; Sariaslan, Sharp, D'Onofrio, Larsson, & Fazel, 2016; Todis & Glang, 2008; Todis, Glang, Bullis, Ettel, & Hood, 2011). In the long term, young adults who sustain a severe TBI during childhood have fewer close friendships, experience poorer educational outcomes, have higher rates of unemployment or underemployment, and endorse lower quality of life, compared to healthy peers (Anderson, Brown, Newitt, & Hoile, 2009; Cattelani, Lombardi, Brianti, & Mazzucchi, 1998; Todis & Glang, 2008).

Factors that Influence Outcomes

One intriguing facet of pediatric TBI is the heterogeneity in long-term cognitive, emotional, and behavioral outcomes. As noted above in the discussion of TBI incidence and prevalence rates, a number of factors are linked with outcomes, including characteristics of the injured child, demographic and family factors, characteristics of the injury itself, and associated neuroanatomical abnormalities.

Individual Characteristics

The types and severity of long-term consequences of pediatric TBI vary based on characteristics of the individual child or adolescent. One important factor is the age at injury. The impact of acceleration–deceleration on diffuse brain systems (particularly white matter) and the diffuse consequences of secondary injuries contribute to poorer outcomes among those who sustain injuries at a younger age (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Barnes, Dennis, & Wilkinson, 1999; Dennis, Wilkinson, Koski, & Humphreys, 1995; Ewing-Cobbs et al., 1997; Taylor & Alden, 1997; Verger et al., 2001). Early injury is also linked with poorer distal outcomes like poor employment outcomes (Anderson, Catroppa, Morse, Haritou, & Rosenfeld, 2005; Todis et al., 2011).

An individual's pre-injury functioning can also predict long-term outcomes after TBI. Children with better cognitive functioning prior to an injury preserve more functional capacity afterward (Dennis, 2000; Dennis, Yeates, Taylor, & Fletcher, 2007; Kesler, Adams, Blasey, & Bigler, 2003), consistent with theories of "cognitive reserve." The cognitive reserve hypothesis (Kesler et al., 2003; Stern, 2003) proposes that those with higher pre-morbid functioning have preserved functional capacity after CNS injury due to the brain's ability to make efficient use of existing networks, which has been supported in few available studies of pediatric TBI (Farmer et al., 2002; Fay et al., 2010). Pre-injury behavioral and mental health histories are also important to consider. Children with a pre-injury history of ADHD, learning disability, or mood disorder are at higher risk for long-term morbidities following TBI (Biederman et al., 2015; Haarbauer-Krupa et al., 2017; Kania, Shaikh, White, & Ackerman, 2016; Yeates et al., 2005).

Socioeconomic and Family Characteristics

Socioeconomic status and a family's overall functioning also play a role in the emergence and persistence of long-term morbidities after TBI (Taylor et al., 2002, 2010; Taylor, Swartwout, et al., 2008; Yeates et al., 2012; Yeates & Taylor, 1997). Some of this influence is likely due to increased support during the acute recovery and rehabilitation process, which can increase the effectiveness of these interventions (Sander, Maestas, Sherer, Malec, & Nakase-Richardson, 2012). These family-level factors and social-environmental influences continue to play a role in

influencing outcomes over the long run as well (Gerring & Wade, 2012). Socioeconomically, children who live in households with fewer resources or economic disadvantage tend to have worse cognitive and academic outcomes, particularly after severe injury (Taylor et al., 2002; Yeates, Taylor, Walz, Stancin, & Wade, 2010). Low economic resources can also impact social and behavioral outcomes. Children may have less access to or ability to consistently seek out specialized care. They may be less able to manage appointments, have limited access to transportation, or have struggle with the addition of a significant health event to an already chaotic or stressed home environment (Johnstone, Nossaman, Schopp, Holmquist, & Rupright, 2002; Lishner, Levine, & Patrick, 1996; Marcin et al., 2004; Mayer, Slifkin, & Skinner, 2005). Family socioeconomic disadvantage, when combined with severe TBI, is associated with the poorest outcomes for children and adolescents.

Also within the home, the level of parental responsiveness and warmth and the types of discipline used predict a child's recovery, especially behavioral outcomes (Wade, Cassedy, Walz, Taylor, Stancin, & Yeates, 2011; Wade et al., 2002). As mentioned above, a TBI is an unexpected and significant health event that can significantly alter typical family functioning, roles, and responsibilities. A parent's absence from work may introduce additional financial strain, although it is sometimes necessary to provide the level of supervision required after a significant injury. Parents may change their parenting or disciplinary approach after TBI (Root et al., 2016), and this may or may not extend to other siblings, possibly introducing an additional basis for family conflict. Overall, pediatric TBI introduces numerous new sources of stress to a family system that can disrupt marital relationships (Wade, Taylor, Drotar, Stancin, & Yeates, 1996), intensify parental or sibling distress (Jaffe et al., 1992; Rivara et al., 1996; Wade, Taylor, Drotar, Stancin, & Yeates, 1998), and contribute to maladaptive or insufficient coping responses (Wade et al., 2001, 2002; Yeates et al., 2002). These factors are strongly related to a child's recovery after TBI, especially emotional and behavioral outcomes (Rivara et al., 1996; Taylor et al., 1999, 2002; Wade, Carey, & Wolfe, 2006; Yeates et al., 2010).

Injury Characteristics

Injury severity is one of the most commonly cited predictors of long-term outcomes after TBI. Overall, the more severe an injury, the worse the neuropsychological and behavioral outcomes (Anderson et al., 2006; Fletcher, Ewing-Cobbs, Francis, & Levin, 1995; Schwartz et al., 2003; Taylor, Yeates, et al., 2008; Taylor et al., 2001). Individual studies vary in how injury severity is assessed. As mentioned previously, the GCS score is one common measure that reflects a child or adolescent's level of consciousness and responsiveness after TBI. It has been linked with both cognitive and functional outcome, as well as the extent of long-term cerebral atrophy (Ghosh et al., 2009). Duration of loss of consciousness and duration of posttraumatic

amnesia are other metrics, and while the GCS provides a snapshot of a child's consciousness at a single time point, duration of loss of consciousness, and posttraumatic amnesia reflect rate of recovery over time. These measures generally perform better than static measures (e.g., GCS) in predicting distal outcomes.

Children who sustain non-accidental or abusive head trauma, especially at a younger age, typically demonstrate worse outcomes because of the severity of such injuries. Non-accidental head trauma is more common in infants and very young children. They suffer more extensive secondary injuries, particularly hypoxic injury due to brain swelling, which causes diminished blood circulation in the brain (Ewing-Cobbs, Prasad, Kramer, & Landry, 1999; Hymel, Makoroff, Laskey, Conaway, & Blackman, 2007; Ichord et al., 2007). Unfortunately, children who sustain non-accidental head trauma are also more likely to come from families with higher stress and fewer resources. This confluence of factors contributes to especially high risk for poor outcomes and death in this young population.

Neuroanatomical Abnormalities

As neuroimaging methods have improved, so too has research on the neuroanatomical abnormalities that predict long-term cognitive and behavioral deficits after pediatric TBI. After injury, CT is typically the modality of choice for initial assessment, as it is readily available, fast, and effective in detecting abnormalities that require emergent intervention (e.g., mass lesions). That said, CT is not especially sensitive to subtler neuroanatomical abnormalities that are common after TBI; magnetic resonance imaging (MRI) has eclipsed CT as the method of choice for detecting smaller lesions, microstructural changes, and cortical atrophy and is often conducted when neurological findings are present but are unexplained by CT (Duckworth & Stevens, 2010).

Individual studies have linked diminished functional connectivity in the corticostriatal motor network and poor response inhibition (Stephens et al., 2017), as well as relatively greater activation in the right frontal and parietal regions during the completion of an inhibitory control paradigm during functional MRI (Tlustos et al., 2011). Others have focused on the assessment of volumetric changes (e.g., lesion volume), using a variety of neuroimaging techniques. For instance, in one longitudinal study of changes in cortical thickness following moderate to severe TBI, Wilde and colleagues found decreased cortical thickness bilaterally within several frontal regions and the anterior cingulate when compared to children who had sustained an orthopedic injury, at 3 months post-injury (Wilde, Merkley, Bigler, Max, Schmidt, Ayoub, et al., 2012). Fifteen months later, persistent decreased cortical thickness was observed in the bilateral frontal, fusiform, and lingual regions, and progressive cortical thinning was apparent in other bilateral frontal, fusiform, and left parietal regions. Longitudinal changes in cortical thickness within the left frontal, right medial frontal, and anterior cingulate regions were significantly correlated

with the aspects of day-to-day executive function (Wilde, Merkley, et al., 2012). Similarly, children with increased intracranial pressure after TBI displayed reduced white and gray matter volumes in the frontal, callosal, and subcortical regions that corresponded with poorer performance on measures of working memory, decision-making, and impulsivity (Slawik et al., 2009).

Much existing research in pediatric TBI focuses on the assessment of white matter pathways, with the goal of quantifying the degree of diffuse traumatic or axonal injury and its association with cognitive outcomes. Diffuse damage to white matter pathways is generally considered to be a major contributor to the cognitive impairments faced by children after TBI (Arfanakis et al., 2002). As noted above, DTI is the most common sequence used to assess white matter integrity. A recent meta-analysis synthesized the findings of 14 individual studies that used DTI to examine the link between white matter integrity and cognitive sequelae of pediatric TBI (Roberts, Mathias, & Rose, 2016). Shortly after injury, increases in FA and decreases in apparent diffusion coefficient (ADC; reflects the rate of diffusion without respect to direction) were associated with poorer performance on the measures of executive function and memory; associations with other cognitive domains (e.g., attention, information processing, construction, motor function) were consistently in the expected direction with moderate to large effect sizes, but statistical significance was sometimes precluded by small samples. At longer intervals post-injury, as expected, effects were in the opposite direction; in other words, decreases in FA were related to poorer performance on measures of academic achievement, attention, information processing, construction, overall cognition, memory, and motor function, with effects in the moderate to large range (Roberts et al., 2016). The change in direction of association likely reflects the early effects of brain swelling and edema, in contrast to the later effects of white matter damage.

Neuroanatomical abnormalities have also been linked to social impairments, particularly theory of mind and other aspects of social cognition. Lesion burden in the mirror neuron empathy network (i.e., inferior frontal, inferior parietal, and premotor regions) and diminished volume in the posterior cingulate/retrosplenial cortex and hippocampal formation were associated with poorer performance on a conative theory of mind task that required appreciation of complex social overtures like irony and empathy (Dennis et al., 2013). Susceptibility weighted imaging, which is particularly sensitive to the microhemorrhagic lesions that accompany diffuse axonal injury, has also been associated with poorer performance on a cognitive theory of mind task (Ryan et al., 2015a, 2015b). In contrast, others have found inconsistent relationships among lesion burden or focal pathology and social deficits (Bigler et al., 2013), illustrating the heterogeneity and complexity of TBI in children. Little research has examined the link between neuroanatomical abnormalities and emotional and behavioral outcomes, although isolated studies have linked white matter integrity within the uncinate fasciculus to emotional and behavioral regulation (Johnson et al., 2011) and white matter integrity within the cingulum, genu of the corpus callosum, inferior longitudinal fasciculus, and inferior fronto-occipital fasciculus to emotional processing (Schmidt et al., 2013).

Cognitive Rehabilitation Techniques and Findings

As appreciation of the long-term morbidities of pediatric TBI has increased, so too have efforts to develop and implement interventions to address them. The amount of empirical evidence supporting interventions for neurocognitive deficits after pediatric TBI continues to lag substantially behind that for adult TBI, but efforts have increased over the past two decades. Existing interventions largely fall under the umbrella of "cognitive rehabilitation," primarily targeting deficits in neurocognitive functions such as memory, attention, and executive functions. Many of these studies assess the impact of interventions using behavioral or cognitive outcome measures, and very few examine changes in aspects of structural or functional brain integrity post-intervention.

Cognitive and Behavioral Outcomes

Typical approaches to cognitive rehabilitation involve intensive, hands-on, or computerized training in specific skill areas. For example, Galbiati et al. (2009) assessed the benefits of an attention-training program for children who were being discharged from inpatient rehabilitation 6–10 months after sustaining a severe TBI. Children participated in intensive training with a therapist four times per week for 45 min per session, over 6 months. Individual sessions included 15 min of tabletop tasks that targeted selective, focused, sustained, and divided attention. For example, during one task, a participant might be presented with a vignette or picture depicting a situation where attention would be challenging; the participant then responded to open- or closed-ended questions to generate options or suggestions to manage attention in that context. The remaining 30 min was spent on computer-based activities (i.e., RehaCom (Schuhfried, 1996) or Attenzione e Concentrazione (Di Nuovo, 2001), based on participant age) that targeted vigilance, attention/concentration, and response behavior. Post-intervention, the children who received the intervention improved significantly on a computerized attention task (CPT-II), as well as on ratings of adaptive behavior provided by parents, and showed significantly more improvement on these measures than did a control group (Galbiati et al., 2009). While this certainly suggests some promise for a combination of massed practice and teaching of metacognitive skills, the intervention was very time-intensive, and the extent to which the testing and parent-reported improvements would transfer to observable behavior is unknown.

A similar pattern of results was observed in a pilot case series involving another computerized cognitive training program, Brain Games (Apple, SpriteKit). After 8 weeks of training, five participants with moderate to severe TBI improved on a flanker and computerized performance test, as well as on parent-reported BRIEF measures; however, no evidence for far transfer of treatment effects was provided (Verhelst, Vander Linden, Vingerhoets, & Caeyenberghs, 2017). Another pilot study

assessed the effectiveness of the Attention Improvement Management (AIM) program, which purports to improve attention and executive function skills (Treble-Barna, Sohlberg, Harn, & Wade, 2016). Thirteen children with complicated mild to severe TBI and a comparison group of 11 healthy children participated in the intervention, which included 10 weeks of face-to-face sessions with a clinician. During sessions, attention-training tasks were selected through a computerized portal, and clinicians coached participants on how different metacognitive strategies could be applied in real-life situations. Between sessions, the participants were instructed to practice the computerized tasks at home 2–4 times per week. Children with TBI improved more than healthy children on a measure of sustained attention after the intervention, as well as on parent-reported executive function. That said, several measures of attention and executive function remained unchanged post-intervention, and the program had considerable attrition and low adherence to the ideal schedule of home-based practice.

Other intervention approaches first trialed in other pediatric populations have been more recently used with children following TBI. Cogmed™ (Pearson Education, Inc.), for instance, is an adaptive working memory training program that has been used in a variety of populations of children with neurodevelopmental (e.g., learning disabilities, ADHD) and acquired neurocognitive deficits. Recently, children with moderate to severe TBI were invited to participate in a Cogmed™ intervention trial that involved 30–40 min sessions, 5 days per week, for 5 weeks (Phillips et al., 2016). As mentioned above, this program is adaptive, so individual trials increase or decrease in difficulty to match a child's performance. Though this intervention was completed at home, children were supervised by parents and had a weekly check-in with a certified training coach. As a control arm, children in an active placebo group also completed the Cogmed™ program, but used a version that was not adaptive and remained at a low difficulty level across the entire intervention. Overall, children in the adaptive arm showed significantly greater gains than children in the control arm on working memory tasks and in some reading-based academic domains, suggesting some possible transfer to daily academic functioning.

Finally, some investigators have attempted to intervene to address behavior problems that occur after TBI. The most prominent series of studies has examined the impact of several different modalities of family-based problem-solving interventions, which have generally proven efficacious in reducing both behavior problems and deficits in executive function relative to internet-based resources (Kurowski et al., 2013; Wade et al., 2006; Wade, Walz, Carey, McMullen, Cass, Mahone, & Yeates, 2011; Wade et al., 2010; Wade, Stancin, Kirkwood, & Brown, 2014; Wade et al., 2015). These findings were most impressive for older youth and those from lower-income families (Kurowski et al., 2013; Wade et al., 2006). Continuing efforts from this research group includes assessment of the extent to which telehealth provision of these interventions is feasible, acceptable, and effective in addressing neurocognitive and behavioral deficits after TBI when compared with internet-based resources or traditional face-to-face intervention.

In a recent review of interventions targeting attention problems, Backeljauw and Kurowski (2014) outlined a number of issues that limit the impact of this growing

body of research. In particular, they noted a lack of consistent outcome measures across studies, as well as a lack of measures that assess changes in day-to-day functioning, which are needed to assess the broader effectiveness of interventions. Furthermore, existing research is so limited that it precludes efforts to prescribe individualized or tailored empirically based interventions. Many individual studies have small samples, occur at a single center, and are unable to delineate findings based on individual (e.g., gender) or injury-related factors (e.g., severity, time since injury). Others pool samples of children with "acquired brain injury" (ABI), obscuring specific effects for children with TBI relative to children with other forms of ABI.

These challenges affect both qualitative reviews and quantitative meta-analysis (Robinson, Kaizar, Catroppa, Godfrey, & Yeates, 2014). For instance, in a recent meta-analysis of randomized controlled trials of cognitive interventions for central nervous system and neurodevelopmental disorders, large, significant, and positive treatment effects were found for attention, working memory, and memory tasks, while small but significant effects were found for academic achievement and behavioral rating scales. However, effects across domains displayed notable heterogeneity, including across diagnostic groups, although the number of available studies and the small sample sizes in those studies did not allow for more nuanced analysis of subgroups (Robinson, Kaizar, et al., 2014). In general, less evidence was found for the generalization of intervention to everyday behavior than for immediate transfer to cognitive tasks. Heterogeneity in intervention methods, outcome measures, and participant groups diminishes the likelihood that existing research can meaningfully inform targeted or precision-based implementation, despite a strong desire to be able to do so within the broader field. Overall, while this is an important area of research that has the potential to positively impact children and their families, substantial work remains to be done.

Neuroimaging Outcomes

Surprisingly few intervention studies have assessed treatment-related changes in structural or functional brain integrity after pediatric TBI, and most of the existing studies do not focus on cognitive rehabilitation programs. For instance, structural connectivity analysis has been used to assess changes in response to an aerobic training program for adolescents with persistent post-concussive symptoms, with some notable increases in global brain efficiency and decreases in normalized characteristic path length following the intervention (Yuan, Wade, Quatman-Yates, Hugentobler, Gubanich, & Kurowski, 2017). DTI has also been used to assess changes in white matter integrity in the cerebellum in response to postural control/balance training following pediatric TBI. Participants who completed 8 weeks of a balance training using PC-based portable balancers showed increased mean diffusivity in the inferior cerebellar peduncle; increases in FA in the inferior cerebellar peduncle were also associated with improvement on balance testing (Drijkoningen et al., 2015).

We could identify only one study that has assessed structural and functional changes in brain integrity following cognitive rehabilitation. Yuan and colleagues (2017) evaluated the impact of participation in the AIM program described above (Treble-Barna et al., 2016) using graph theory analysis to analyze global and regional brain networks. They found significantly higher small-worldness in the TBI group relative to the healthy control group at baseline, which indicates that the networks in the TBI group have more closely connected, short-distance connections, rather than long-distance connections, possibly reflecting greater reliance on segregated versus integrated brain networks. Following the AIM intervention, children in the TBI group showed reduced small-worldness and normalized clustering coefficient at follow-up. Improved network characteristics, including mean local efficiency and normalized characteristic path length, were associated with improvements in verbal inhibition, parent- and child-reported executive function, and sustained attention (Yuan, Treble-Barna, Sohlberg, Harn, & Wade, 2017).

Practice Considerations

Though currently quite limited, the existing body of research should begin to inform clinical practice and research, given the public health significance of pediatric TBI. Furthermore, ongoing and future research should continue to strengthen and refine our approaches to managing the neurocognitive and behavioral sequelae of these injuries.

Considerations for Assessment

One key role for practicing clinicians, particularly neuropsychologists, is providing rigorous assessment to help develop educational accommodations and behavioral and emotional supports for children who have sustained TBI. When approaching assessment, a variety of risk factors for poor outcome should be considered, including demographic and socioeconomic disadvantage, worse pre-injury functioning, and more severe injury. These should be weighed against factors that might promote more resilience, such as greater cognitive reserve or a high degree of family support. Assessments should target the domains most vulnerable to TBI (e.g., executive functioning, memory, processing speed), as well as skills that are less vulnerable (e.g., basic reading skills) to identify areas of strength and weakness.

Using what is known about the evolution of deficits across time after injury, clinicians can help families and educators anticipate potential future challenges as a child continues their recovery, and as he or she approaches major developmental milestones. While some challenges may be cognitive, the social, emotional, and behavioral morbidities that occur after pediatric TBI also must be remembered, and

clinicians must provide guidance and recommendations that address each unique aspect of a child's overall functioning.

Considerations for Treatment

As discussed above, strong empirical support for any single type of treatment (whether cognitive or behavioral) for the outcomes of childhood TBI is lacking. Across different specific intervention programs, some support can be found for at least some potential benefit of cognitive interventions that target skills like working memory and attention, at least in terms of near transfer, and additional research is underway. Support for transfer to more distal skills and assessment of whether benefits are sustained over time is even more limited. Given the demanding schedule of cognitive interventions, the benefits of these treatments should be weighed against other competing demands, particularly during acute recovery.

Even less research has focused on the treatment programs for the emotional and behavioral sequelae of pediatric TBI. However, this is an area of significant interest and growth, including outside of the realm of "cognitive rehabilitation." For instance, a recent study of children and adolescents with mild TBI and prolonged post-concussive symptoms found significant symptom improvement and increased quality of life ratings in response to participation in a cognitive behavioral intervention (McNally et al., 2018). The intervention focused on psychoeducation about mild TBI and typical outcomes, activity and sleep scheduling, relaxation and biofeedback training, and cognitive restructuring. Though quite preliminary, the development of these types of program, and "manualization" and dissemination, may prove highly beneficial for youth with TBI.

Considerations for Research

Finally, considerable room for growth exists in how researchers develop, implement, and assess cognitive rehabilitation programs. As mentioned above, many existing studies are limited by small and heterogeneous samples that limit the ability to examine mediators or moderators of treatment effects. In a broader healthcare system that is moving toward precision medicine, this gap is highly problematic. Increasing multicenter collaborations may help address this issue. When conducting pilot or single-center studies, moving toward consistency in outcome measures would at least allow for side-by-side or meta-analytic review of findings, perhaps guided by the NIH Common Data Elements Project (McCauley et al., 2012). Though challenging, randomized and controlled trials are especially useful in determining the efficacy, as well as the effectiveness, of interventions.

As interventions continue to be developed and tested, researchers should also incorporate neuroimaging methods when assessing their efficacy. While we believe

this is true across different types of brain injury, use of neuroimaging markers in pediatrics can elucidate how TBI operates within the context of neurodevelopment, and how these dynamic processes can validate treatment, inform risk stratification, and foster and monitor recovery. Mounting evidence supports these methods, particularly DTI and measures of network connectivity, as sensitive to the structural and functional consequences of TBI on the brain. If these methods prove useful in predicting or assessing response to treatment, our understanding of the mechanisms supporting functional improvements in this population would be greatly increased. Furthermore, though neuroimaging is costly, and some methods are not readily available at some institutions, the discovery that a particular sequence or set of sequences is predictive of or sensitive to recovery could inform the clinical management of acute injury, monitoring over time, and the implementation of individualized treatment for children and adolescents with TBI.

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